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THEORIES OF ASTIGMATISM

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Abstract

Patients with astigmatism are encountered virtually every day by practicing clinicians. This paper consists of an extensive literature review of the theories of astigmatism development, from both a mechanical and functional aspect. It is a goal of this work to present a broad view of the field which can be useful in day to day encounters with astigmatic patients.

Introduction

The changes in astigmatism which come about as a result of aging and development are well documented, and generally well accepted by the ophthalmic community. Although one of the original innovators of much ophthalmic knowlege, Donders (1), maintains that there is no change throughout life, most research from that time to the present demonstrates a definite series of predictable changes. Extensive research with subjects aged birth to over 90 years, along with the practical, everyday observations of clinicians, has allowed the idea of astigmatism development to become well established.

In addition to the physical aspects of astigmatism development, many researchers have investigated changes in astigmatism relating to the functional use of the eyes. Much of this research indicates that a person's astigmatic status can change in relation to how the visual system is used, and in relation to a person's posture and habitual eye movement patterns.

This paper will explore the development, progression, and occasional regression of astigmatic changes from both a physical aspect, and a functional aspect. Armed with this overview, a practicing clinician can then utilize the theories which seem most relevant for an individual

patient, and prescribe (or not prescribe) lenses or therapy accordingly.

Development and Progression

Infants and children show some very interesting astigmatic changes as they develop. Generally these changes are of a much larger magnitude than any astigmatic changes which occur later in life. Considering infants from birth to 12 months, 45% have at least 1.00 diopter of astigmatism (2), and 63% have at least .75 diopters of astigmatism (3). Astigmatic errors of this amount are 5-10 times that reported for older children and adults (2).

Using the technique of dark room retinoscopy, Mohindra (2) has broken down the first year of life into several stages. The incidence of astigmatism is low for the first ten weeks of life, highest for ages 11-20 weeks, and lower after 20 weeks. By age 41-50 weeks, most infants have less than 2.00 diopters of astigmatism . Although the validity of dark room retinoscopy has been questioned with regard to determining spherical refractive error in infants (4), the technique to determine cylindrical values is quite valid (5). Throughout the first year, the axes are 70-80% horizontal/vertical, with approximately equal amounts of With-the-Rule (WTR) and Against-the-Rule (ATR).

Studies of children (up to 6 to 8 years) also demonstrate definite trends in astigmatism development. The major trend through this time is a reduction of both WTR and ATR astigmatism. By age 6, 81% of children have less than .25 diopters of astigmatism (6).

From birth to age 3 years, there is no significant difference in the amount of WTR vs. ATR. However, ages 3 to 4 1/2 years show a significantly greater amount of ATR, and ages 4 1/2 to 6 years show more WTR (7). This tendency for greater WTR remains through the school years, and throughout pre-presbyopic adulthood.

A non-astigmatic infant is unlikely to develop astigmatism before the school years (7). During the school years, 75% of eyes change less than .25 diopters, and only 2% change more than .75 Diopters (6).

Changes in astigmatism again demonstrate a trend at about the time of the onset of presbyopia (about age 40). From this time onward, the general population shows a shift from predominantly WTR to predominately ATR astigmatism. This change occurs at an average rate of .25 diopters /ten years, and the average amount changes from .25 WTR at age 40, to .75 ATR at age 80(8). This is apparently a steady, continuous change, as no one age group shows a faster acceleration of astigmatic changes.

Baldwin (9) has conducted a longitudinal study of patient's records spanning 40 years. These patients had records from before age 30 and after age 70. Again the trend toward increased ATR astigmatism is apparent. According to Baldwin, "...the most consistent contribution to total astigmatic change during this age period is steepening of the horizontal corneal meridian".

An important issue must be addressed regarding the above studies. In two of the studies on infants and children (2,7), 95% to 97% of the subjects were caucasian. It is possible to assume that the other studies cited involved predominantly white subjects. There is research to indicate that different races show different trends regarding astigmatic development (10,11). Therefore the above generalizations regarding the changes in astigmatism with aging can be applied with confidence only to caucasians.

There has been little work done examining oblique astigmatism (broadly defined as astigmatism in which the principal meridians are greater than 30 degrees away from horizontal or vertical). It has been found that only a small proportion (<20%) of people demonstrate oblique axes (2,3,7), and these data are generally omitted from studies of astigmatism.

Physical Aspects

It is generally agreed that a refractive error occurs when one or more of the optical components of the eye are mismatched in relation to the others. The important components to consider are: corneal power and shape, lens power and shape, depth of the anterior chamber, depth of the vitreous chamber, and overall length of the eye. Refractively, the most powerful of these components is the front surface of the cornea, and it is this component which is most important when considering astigmatism.

The cornea is a viscoelastic tissue which is greatly influenced by the tissues which support it. Intraocular pressure, lid pressure, the effects of the extraocular muscles, etc. all have some effect on the globe, and thereby all directly or indirectly influence the corneal shape.

Lid tension and position are two factors which have long been associated with astigmatism. In eyes initially presenting with greater than .625 diopters WTR astigmatism, lifting the lids away from the globe will significantly reduce the amount of corneal astigmatism. (i.e., there is for this group an average shift of .611 diopters toward ATR) (12). Subjects with initial astigmatic measurements of <.625 diopters WTR showed corneal changes in either direction (more WTR or ATR).

Surprisingly, the major factor influencing this change was an increase in power of the horizontal meridian. One may have suspected the opposite, i.e., a decrease in the power of the vertical meridian with the lifting of the lids.

Grey and Yap (13) have demonstrated that 80% of subjects show an increase in WTR astigmatism with voluntarily narrowed lids. However, the remaining 20% showed an ATR shift. No mechanism is proposed to explain why some subjects responded with an ATR shift. The most surprising finding in this study involves the amount of astigmatic change (the median change was 1.25 to 1.50 D.). This indicates that in adults the cornea is quite flexible in response to outside influences (14).

Other physical factors can influence the amount of astigmatism. Firmly retracting the lids, digital pressure on the globe, pulling of the lids temporally, and rubbing the eyes have all been shown to induce >.50 diopters of corneal astigmatism (15). It is interesting to note that in this study by Mandell, of the seven subjects, only two demonstrated the increased astigmatism due to digital pressure, lid retraction and lid pulling, and the astigmatism due to eye rubbing has been attributed to a disruption of the epithelium or tear layer. Does this indicate a predisposition towards a more malleable cornea in these two subjects?

Unfortunately Mandell does not address this possibility in his study.

It has generally been found that the ciliary muscle action during accommodation does not affect the corneal curvature (15,16). It is possible however, that the act of accommodating may induce astigmatism within the lens. In a review of the literature of astigmatic accommodation, Brzezinski (17) cites 19 articles which address either a sector contraction of the ciliary muscle causing a lenticular astigmatism, or the induced astigmatic optical effects manifested as a slightly tilted lens accommodates. Of these 19 articles, 14 support the possibility of lenticular astigmatism induced by accommodation.

Some very interesting work was done by Fairmaid (16) regarding the effects of convergence upon corneal astigmatism. The shift towards WTR astigmatism measured by Fairmaid was so small he claimed it "clinically insignificant". However, the data from 58 subjects demonstrates certain trends which are noteworthy. 56 of the 58 subjects showed changes towards WTR astigmatism with convergence. The average shift in corneal readings was a .022 mm increase in radius of the horizontal meridian, and a .009 mm decrease in radius of the vertical meridian. In terms of clinical practice, this small amount of change is certainly insignificant, however, one must accept it's existence and speculate on the effect of

this influence acting on a cornea every time a person converges throughout their entire life. Perhaps a habitual asymmetric convergence posture could manifest itself into an anisometropic astigmatism through this mechanism? These ideas will be discussed later in this paper.

Functional Aspects

It is relatively easy to discuss astigmatism as a physical entity. The optical characteristics of an astigmatic eye can be described and measured, and the influence of various extraocular components and their effect on the eye's shape have already been discussed.

There exists however, another aspect of astigmatism to be addressed. A person's physical make-up is not determined solely by heredity, but also by environment. The same holds true for a person's vision. A person's behavioral interaction with the input to his/her visual system will have an effect upon the structures which comprise the visual system. This organism/enviornment adaptability takes many forms and operates to many degrees. Following are the thoughts of various researchers as to how environmental interactions can affect a person's astigmatism.

Perhaps the most extensive work done in the field of environmental

influences on the visual system is that of Harmon (18). He has studied extensively the effects of posture, lighting, body orientation, and task orientation on the visual system. He holds that the ability to adapt is inherent in all living tissue. The particles which make up living protoplasm "tend to seek equilibria within themselves and with other substances so as to make for greater stability." When an external stressor is applied to a structure, the protoplasm of the structure will attempt to re-establish an equilibrium of less energy (i.e., a more stable equilibrium). Intensity and duration of the stressor, combined with the initial stability of the structure, will determine the amount and permanence of any induced structural change.

Entire systems are affected in a similar way; however it is generally acknowledged that a system is a sum of it's parts. Each part cannot perform without every other part. The entire system (in this case the visual system, from cornea to cortex) operates only because of an orchestrated coordination of it's parts. When an external stressor is now applied to the system, any adaptation by one part will have some effect upon the operation of the entire system. For example, a child may perform all his/her near work with a habitual head tilt. This causes an alteration of input to the visual system which must be dealt with at some level

(initially probably within the perceptual realm of the cortex). As this alteration becomes habitual (i.e., the duration of the applied stressor increases) the effects become more profound. In order to maintain the entire system at the lowest (most stable) energy equilibrium, a structural change may begin to occur, perhaps in the manner of a corneal curvature change.

Forrest (19,20,21) has proposed a model in which changes in a person's astigmatic status are a function of the person's visual scan, head scan, and head posture mannerisms. Visual scan refers to eye movement with no accompanying head movement, head scan refers to yoked eye and head movement, and head posture refers to head position relative to gravity.

The key to the interrelationship of these factors appears to be freedom of movement. For example, if a person tends to use a visual eye scan pattern when viewing in one meridian, and a head scan pattern when viewing in the meridian 90 degrees away, an astigmatism will develop with its axis oriented in the meridian of greatest eye scan. Therefore, a WTR astigmatism appears to relate to a greater preference for eye scanning in the 180 meridian.

Forrest has performed three studies to support his theory. The first

two involve studying subjects' habits, postures, and tasks, and predicting their eye/head scan preference on the basis of their astigmatic correction. The third was a clinical study in which visual scanning therapy reduced the astigmatism in an experimental group (n=45) as compared to a control group (n=45) by an average of over .40 diopters (subjective refraction, $p<.0001$).

This work is similar to that done by Childress et. al. (22) who related the astigmatic error of 69 patients to their occupations. For example, secretaries tended towards WTR astigmatism (presumably due to their predominantly horizontal eye movement patterns), while telephone operators tended toward ATR.

Forrest also examines anisometropic astigmatism as it relates to task orientation. If a person habitually postures him/her self with his/her task to one side, the eye farthest from the task will develop a greater astigmatic error. Perhaps the trend found by Fairmaid (16) (that of a slight corneal curvature change with convergence) is one of the inciting mechanisms for this anisometropia.

Harris (23) has examined the visual conditions of symphony musicians. These musicians spend years of their lives practicing and performing in unusual postures, thereby inducing unusual stresses to

their visual systems. These stresses are reflected in the astigmatic and anisometropic aspects of these performers' visual systems in much the same way as predicted by Forrest. Perhaps the most interesting example of this postural influence occurs in violinists who present with parallel axes at 10 degrees, with the eye furthest from the music stand demonstrating the greater astigmatisn.

Another functional aspect to be considered is that of how low amounts of ATR asigmatism relate to incipient myopia and binocular difficulties. Birnbaum (24) has proposed a theory in which this low ATR astigmatism is an adaptation which reduces near point stress by allowing a greater lag of accommodation, while maintaining resolution for the primarily vertically oriented characters of the English alphabet. This adaptation reduces the stress on the accommodative system, while only minimally affecting distance acuity (as compared to myopia). Birnbaum recommends clinicians consider the judicious use of plus lenses for patients exhibiting low amounts of ATR astigmatism of recent orgin. This reduction of near demand may reduce the early astigmatism and may slow any subsequent myopia development.

Discussion

In reviewing the work of these various researchers, an interesting correlation begins to present itself. Ludlam (25) has noted that persons who have reading difficulties related to visual problems tend to be "head movers". Their binocular inefficiency causes them to skip lines and lose their place easily, therefore they move their whole head, while keeping their eyes relatively fixed straight ahead. Forrest's model suggests that the meridian involving restricted eye scan becomes relatively more myopic (i.e., the axis of easiest eye movement corresponds to the axis of the astigmatism). Harris has attributed this to the agonist/antagonist relationship of the extraocular muscles (i.e., there is increased tonus on both the medial and lateral rectus to hold the eyes relatively straight ahead when the eye and head movements are yoked).

When taken together, these theories all tend toward a mechanism for increased ATR astigmatism in response to near stress. In addition Birnbaum's work, which demonstrates how optically, an ATR astigmatism permits a greater lag of acommodation while maintaining resolution of vertically oriented characters, demonstrates a sensory/motor benefit for a person to fall into this mechanism.

Summary

At one extreme, astigmatism may be considered genetically pre-programmed, following an unavoidable progression throughout life. At the other extreme, it may be considered an ultra-sensitive indicator of how a person is using their eyes, wherin the slightest environmental change will cause a measurable astigmatic change. As scientists and clinicians, it is our job to realize that the real answer probably lies somewhere between these extremes. We must approach each patient with an open mind, always ready to utilize the full scope of our training and knowledge.

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